

The Role of Ecotones in Emerging Infectious Diseases

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Abstract: Recognition of the significance of the boundary between ecological systems, often referred to as the ecotone, has a long history in the ecological sciences and in zoonotic disease research. More recent research in landscape ecology has produced an expanded view of ecotones and elaboration of their characteristics and functions in ecosystems. Parallel research on emerging infectious diseases (EIDs) and the causes of increased rates of pathogen transmission, spread, and adaptation suggests a correspondence between ecotonal processes and the ecological and evolutionary processes responsible for zoonotic and vector-borne emerging infections. A review of the literature suggests that ecotones play a role in a number of the most important EIDs. Yet these are the only diseases for which specific landscape ecological information exists in the literature or disease reports. However, the similar disease ecologies of these with about half of the approximately 130 zoonotic EIDs suggests ecotones, particularly their anthropogenic origination or modification, may be generally associated with ecotones and the global trend of increasing EIDs.

Key words: ecotones, EIDs, emerging infectious diseases

INTRODUCTION

Ecotones, the edges or transition zones between two adjacent ecological systems are frequently mentioned in the zoonotic and vector-borne disease literature. They are used to describe field study or sampling locations (Bennett et al., 1999; Townsend et al., 2003; Eisen et al., 2004) and features of possible surveillance value using remote sensing (Wilson, 2002b). Some recent field studies have suggested ecotones as habitats where rates of infectious parasitic diseases in wildlife are high and may increase due to climate-driven shifts in ecotones (Svobodova et al., 2004). Studies in Europe and North America have shown tick vectors of a number of significant zoonotic infectious diseases are most

abundant in these ecotones (Goddard, 1997; Lindstrom and Jaenson, 2003; Pietzsch et al., 2005). Mosquitoes responsible for the transmission of encephalitis virus have been found to congregate at ecotones (Lothrop et al., 2002), and populations of competent rodent reservoirs (white-footed mouse) for Lyme disease preferentially inhabit ecotones (LoGiudice et al., 2003). Ecotones have been identified as potentially important for some eukaryotic parasites in terms of host seeking (Burg, 2001) and host switching (Hoberg et al., 2002). The pathogen's themselves may concentrate in their host populations at ecotones as is apparent for some arboviruses (Yuill, 1986). Bradley (2004) briefly summarized the history of ecotones in epidemiology with regard to their significance in providing specialized habitat for hosts or vectors, as the interface between human settlements and natural ecosystems, and where humans come in contact with a "new" ecosystem. However, his

focus was on an analogous concept he coined “chronotones,” and their potential for contributing to understanding disease outbreaks and control measures targeting environmental factors. Yet the role of ecotones as a factor contributing to the global EID phenomenon has not been critically examined taking into account the synthesis based on a re-examination of ecotones based by landscape ecologists (Holland et al., 1991; Hansen and Di Castri, 1992; Risser, 1995). In this article, we briefly summarize this expanded view of ecotones and consider the evidence based on a review of the literature and EID reports for the role of ecotones in infectious disease emergence on this basis.

EXPANDED VIEW OF ECOTONES

Ecotones were originally described as specialized wildlife habitat represented by readily identifiable edges or transitions zones between major vegetation cover types (Clements, 1905; Leopold, 1933; Odum, 1971). They were viewed as relatively static zones and landscape boundaries favored as habitat by some species and crossed by other species encroaching “alien” habitat or ecosystems. In the new synthesis (Holland et al., 1991; Hansen and Di Castri, 1992; Risser, 1995), ecotones are described as dynamic and more complex constituents of ecological systems that influence biodiversity and ecosystem function disproportionately to their geographic extent, and all spatial scales. This new synthetic view has been substantiated by theoretical and empirical studies (Smith et al., 1997; Fagan et al., 1999).

The principle conclusion of this body of research is that ecotones can now be described as areas where biophysical factors, biological activity, and ecological evolutionary processes are concentrated and intensified. This includes biological productivity, genetic diversification, evolutionary adaptation, interspecific interactions, and the aggregation and movement of organisms, nutrients, and other materials (Risser, 1995), although a single ecotone may not exhibit all these characteristics (Walker et al., 2003). Another dimension of this rethinking of ecotones is their description as representing a spatial hierarchy (Gosz, 1991, 1993). Thus, an ecotone can be considered the boundary at an individual plant’s edge or a small vegetation patch (on the scale of meters or hectares) to a landscape type (on the scale of 10^4 to 10^6 hectares) or that determined by the transition zone between two biomes (a regional scale of 10^3 + Km²). This biome, or regional scale ecotone con-

sists of a landscape “mosaic” of patches varying in type, number, size, inter-patch distance, and juxtaposition. Its ecotone characteristics reflect a spectrum of patch ecotonal and inter-patch processes depending on the microclimate, soil chemistry, flora and fauna, as well as larger scale abiotic and biotic factors, including human influences across all of these scales.

It follows that ecotones include zones of interaction where large-scale land use change produces moving “fronts” where human settlements and accompanying cropland and pasture expand into relatively intact natural ecosystems. Such ecotones now dominate much of the geography of the world’s tropical developing regions where land use change and forest conversion has been occurring at historically unprecedented rates in the past three decades (Meyer and Turner, 1994; Lambin et al., 2003). Human-created ecotones now extend across entire regions, superimposed on and expanding into natural ecotones, as well as extending deep into formerly intact blocks of continuous forest. Much of the formerly huge block of tropical rain forest extending across equatorial, central, and East Africa has been eroded to a point where all but the core area in central Africa can be described as basically a regional scale ecotone. Population growth and pressure on natural resources has similarly transformed Southeast Asia in recent decades such that zones representing ecotones are now far more extensive than intact forest blocks. This may limit the original utility of the term (edge habitat has become virtually ubiquitous) on this scale. But it emphasizes how processes formerly constrained geographically are now widespread.

ECOTONAL PROCESSES AND THE DISEASE EMERGENCE PROCESS

Dzszak et al. (2000) described how most emerging infectious diseases exist within a host–parasite ecological continuum, while Patz et al. (2000) summarize the types of environmental changes and conditions known to have contributed to emerging zoonotic infectious diseases. These authors and others (e.g., Lafferty and Kuris, 1999; Wilcox and Colwell, 2005; Wilcox and Gubler, 2005) have suggested how parasite–vector–host relationships can be distorted by anthropogenic landscape alterations that contribute to changes in vector breeding conditions in non-human and human host populations, resulting in shifts in host–vector–parasite relationships. Kapan et al. (2006)

Table 1. Correspondence of Ecotone Characteristics and Processes with Ecological Evolutionary and Host–Vector–Parasite Changes Associated with Zoonotic Disease Emergence

Biophysical factors reportedly elevated in ecotones	Ecological or evolutionary condition or change	Host–Vector–Parasite (pathogen) shifts or change producing disease emergence
1. Species richness and density	Increased frequency of novel species contact	Increased opportunity for host-switching by pathogens
2. Genetic variability and diversity	Increased opportunity for genetic exchange, genetic novelty	Increased rates of evolutionary change and adaptation
3. Biological productivity	Increased population growth and turnover rates	Increased frequency of breaching of critical thresholds for pathogen/infection growth and persistence in host population
4. Cross- and along-boundary flows of energy, materials, and organisms	Dispersal and regulation of movement and flows of species, water, and materials	Increased opportunity for pathogen and host dispersal via environmental media (water, air, soil/sediment)
5. Environmental variability and gradients; habitat heterogeneity	Spatial and temporal environmental variation in biotic and abiotic factors	Reinforcing of biophysical factors 1, 2, and 3

describe this in detail for avian influenza (H5N1), describing how microscale ecological evolutionary and macroscale social ecological changes may be linked.

As shown in Table 1, the biophysical factors, biological activity, and ecological evolutionary processes described by ecologists and evolutionary biologists as characteristic of, and concentrated and intensified in, ecotones are largely the same factors thought to contribute to the shifts or changes in hosts, vectors, or parasites (pathogens) that produce disease emergence. These dynamics are associated with land cover/use change and the changing nature of the land–water interface, where parasite and host switches can occur as either “spillover,” cross-species transmissions, or introductions/extensions of geographic range into new or changed habitats (e.g., Patz et al., 2004). Anthropogenic influences reflected in land use/cover change can be described as effectively increasing ecotonal processes by increasing their geographic extent and overlap. The result is often in the intersection of multiple natural and anthropogenic types of ecotones.

Human activity has resulted in the spatial hierarchy of “ecotones within ecotones” being elaborated across scales from that of local habitat patch to the biome, produced by different combinations of natural and anthropogenic habitat and ecosystem types. For example, at the local to landscape scale, these include forest edge habitat and fragmented forest landscapes, and terrestrial–aquatic ecotones represented by riparian habitat and riverine landscapes, and wetland (fresh water and estuarine). At the

biome scale, these primarily include forest–grassland/tundra and terrestrial–marine (coastal zone) ecotones. Anthropogenic ecotones recognizable at the local to landscape scale include cropland/pasture–natural habitat and settlement–natural habitat, and a combination of these. The mixture of different kinds of cropland, pastureland, and natural habitat, with different human settlement types (urban, peri-urban, or rural) coalesce into complexes of ecotones on a regional scale.

EMERGING INFECTIOUS DISEASES ASSOCIATED WITH ECOTONES

It follows that ecotones and their associated processes may play a role in the EID phenomenon. Particularly for those zoonotic diseases whose emergence has been adequately documented, there should be a demonstrable association with ecotones and specific ecotonal processes. We attempted to test this hypothesis by searching the peer-reviewed literature for relevant information on the list of 130 zoonotic EIDs assembled by Taylor et al. (2001), recently updated by Woolhouse and Gowtage-Sequeria (2005). We reviewed the readily accessible (electronically searchable) literature on each disease for information describing the locations, ecological circumstances, and factors reported to have contributed to emergence events. Consistent with the definition of an EID (Lederberg et al., 2003), we attempted to determine whether or not the source or appearance of a new or more virulent variant of a pathogen, infection event,

Table 2. Zoonotic Emerging Infectious Diseases (EIDs) Associated with Ecotones

EIDs ^a	Reservoir	Transmission route	Ecotone involvement	Ecologically similar EID (or pathogens causing them) ^b
Viruses				
1. Sin nombre	Rodents	Contact with rodent excretions	Settlement–Natural habitat ^{c,d}	Andes, Junin, Laguna Negra, Lassa, Machupo, Puumala, Sabia, Seoul, Guanarito, Hantaan
2. Yellow fever	Monkeys	Mosquito vector	Settlement–Natural habitat ^c	Dengue, Chikungunya, Zika, O'nyong-nyong
3. Nipah	Bats	Contact with bat excretions	Agriculture–Natural habitat ^{c,d}	Hendra, Menangle, Australian bat lyssavirus, SARS
4. Influenza	Birds, pigs, humans	Direct or aerosolized contact	Settlement–Agriculture–Aquatic habitat ^{c,d,e}	Japanese encephalitis
5. Rabies	Vertebrates	Direct contact	Settlement–Natural habitat ^c	Ebola, Monkeypox, Marburg
Bacteria				
6. Lyme disease	Rodents, deer	Tick vector	Settlement–Natural habitat ^{c,d}	<i>Anaplasma phagocytophila</i> , <i>Ehrlichia chaffeensis</i> , <i>Ehrlichia ewingii</i> , <i>Babesia microti</i> , <i>Rickettsia prowazekii</i>
7. Cholera	Humans, zooplankton, shellfish	Fecal–oral route; contaminated seafood	Terrestrial–Aquatic habitat ^{d,e}	<i>Vibrio parahaemolyticus</i> , <i>Vibrio vulnificus</i>
8. Leptospirosis	Vertebrates	Direct contact	Terrestrial–Aquatic habitat ^{c,d,e}	<i>Giardia duodenalis</i> , <i>Toxoplasma gondii</i> , <i>Cryptosporidium parvum</i> , <i>Campylobacter fetus</i> , <i>Francisella tularensis</i> , <i>Schistosoma</i> spp., <i>Escherichia coli</i>
Protozoa				
9. Malaria ^b	Humans	Mosquito vector	Settlement–Natural habitat ^d	Chagas, Leishmaniasis, <i>Loa loa</i> , <i>Onchocerca volvulus</i> , Oropouche, Mayaro
10. Sleeping sickness	Vertebrates	Tsetse vector	Settlement–Agriculture–Natural habitat ^{c,d}	<i>Echinococcus granulosus</i> , Anthrax, Rift Valley fever, Crimen-Congo hemorrhagic fever, Kyasunur forest virus, <i>Mycobacterium bovis</i>

^aRepresentative references: Anyamba et al., 2001; Ashford, 2000; Barrett and Monath, 2003; Boshell, 1969; Botto et al., 2005; Boussinesq and Cardon, 1997; Carlson et al., 2004; Chevalier et al., 2004; Daszak et al., 2004; de Castro et al., 2006; de La Rocque et al., 2005; de Thoisy et al., 2003; Endy and Nisalak, 2002; Fagbo, 2002; Field et al., 2001; Fouchier et al., 2005; Jackson, 2003; Jenkins et al., 2005; Khodakevich et al., 1987; Le Guenno, 1997; Lipp et al., 2002; LoGiudice et al., 2003; Maupin et al., 1991; McCrae and Kirya, 1982; Mercer and Castillo-Pizango, 2005; Michel, 2002; Moncayo et al., 2004; Morner, 1992; Munga et al., 2006; Olival and Daszak, 2005; Parola et al., 2005; Peiris et al., 1993; Powers et al., 2000; Rodhain, 1980; Rose, 1997; Rotureau, 2006; Steere et al., 2004; Vinetz et al., 2005; Vitor et al., 2006; Walter, 2003; Wanji et al., 2003; Wasteson, 2001; Weaver, 2005; Webster et al., 1992; Wilson, 2002b; Yapi et al., 2005; Zeier et al., 2005.

^bComposed of taxonomically diverse members (e.g., virus, bacteria, protozoa, helminth).

^cEcological processes (see text).

^dMixing of species assemblages.

^eHost–vector hyper-abundance.

^fEnhanced dispersal.

outbreak or epidemic(s), dispersal or geographic spread of a pathogen has been or still is associated with an ecotone. In addition, we attempted to further determine whether specific ecotonal processes are suggested. For some diseases, an association with ecotones is well-known and has been explicitly described (e.g., Yellow fever and Lyme disease). However, neither this terminology nor other technically precise landscape ecological terminology, or technically precise ecological terminology in general is used in the zoonotic disease literature. So whether or not an association with ecotones and specific ecotonal processes exists can only be inferred for many diseases. For most, however, pertinent descriptive information, regardless of its technical precision, currently is lacking altogether.

Table 2 provides a summary of our results including citations of sources representative of the literature for each zoonotic EID where information pertinent to the emergence ecology of a disease and an association with ecotones was found, inferred, or, in a few cases, suggested that further investigation may establish a role for ecotones. Such information, particularly that definitively showing ecotone involvement or not, is generally sparse except for the most thoroughly studied diseases. We found 10 EIDs for which, in our judgment, sufficient information exists and research has been conducted to assess whether or not ecotones contribute to their emergence, at least over a significant part of the diseases' geographic range. Ecotones and ecotonal processes are associated with the emergence of all the viral diseases in Table 2 and one of the bacterial diseases (Lyme disease). For the remaining four diseases, ecotones appear to play a somewhat less important but still significant role. For example, cholera has an oral–fecal transmission mode but the pathogen's dispersal in the environment often involves aquatic–terrestrial ecotonal processes. Also, malaria involves domestic vector-borne transmission in urban environments as well as transmission in rural areas where forest land use change produces more favorable vector habitat along forest edge ecotones contributing to increased vector abundance.

Although information on the landscape ecological circumstances surrounding emergence was found to be sufficient for only these 10 diseases, for each of these, also shown in Table 2, several other diseases are listed whose emergence ecology is apparently similar. For example, like influenza, the emergence of Japanese Encephalitis has involved transmission among birds and pigs (also mosquitoes in the latter) and the spatial overlap of human settlements, agriculture, and natural habitat. They differ in that water-

birds and wetland habitat are involved in former while non-water birds but irrigation systems providing vector habitat apparently have been important in the latter's emergence. For some of these ecologically similar diseases, the indirect evidence of a role for ecotones is reasonably strong; for example, for the waterborne diseases listed as similar to cholera and leptospirosis, for which ecotones defined by terrestrial–aquatic (and marine for cholera) ecotones clearly play a role in emergence. For others, like Ebola, Monkeypox, and Marburg, listed as similar to rabies, the data is very sparse, the evidence is weaker, and the similarity is less obvious. But the accumulating evidence points toward the role of forest clearing, increasing edge habitat, encroaching human settlements, and direct human–wildlife contact.

In addition to identifying the particular type of ecotone, we indicated the ecotone characteristics or ecotonal processes apparently contributing to emergence for the 10 well-studied diseases based on our interpretation of the literature. One or a combination of two or more ecotonal processes shown in Table 2, keyed to table footnotes with c, d, and/or e next to the type of ecotone, indicate the following kinds of processes: (c) Mixing of species assemblages—populations of species that normally are members of distinct ecological communities from different habitats or ecosystems, overlapping in ecotones, providing the opportunity for pathogen spillover. (d) Host–vector hyperabundance—habitat conditions at ecotones providing for unusually high population sizes of actual or potential host or vector species, allowing for the increased potential for pathogens to achieve critical threshold density (i.e., intrinsic rate of infection, $R_0 \geq 1$). (e) Enhanced dispersal—conditions facilitating dispersal at a higher rate or longer distance, resulting from linear habitat defined by habitat edges, such as riverine or gallery forest, and flowing water in streams or rivers themselves.

Of the 10 known ecotone-associated zoonotic EIDs, the role of ecotones is best documented for Yellow fever. Its ecology and epidemiology has been well described since the mid-1950s and it serves as a classic example of a vector-borne EID associated with an ecotone. The virus is maintained endemically among canopy dwelling monkey species and forest mosquito transmission cycles and may emerge in areas in which human settlement has encroached along the forest fringes, and whose proximal habitations or plantations (e.g., banana) may support domestic and peridomestic *Aedes spp.* of mosquitoes. The mixing of densely populated host and vector species facilitates the efficiency

virus transmission, and disease outbreaks frequently occur successively at multiple points along riverine forests. Dengue is a related arboviral disease that has historically shared similar host, vectors, and ecological niche that is now transmissible in the absence of non-human primate cycles. Yet this disease is believed to have originally emerged as a result of the virus' successful shift from forest mosquito species to domestic and peri-domestic mosquito species. Data are insufficient to determine whether or not sylvatic dengue still spills over to peri-domestic transmission cycles. Both of these are examples of spillover from enzootic foci within natural forest ecosystems into proximal human settlements.

Of the other vector-borne EIDs with demonstrable ecotonal associations, Lyme disease is the most thoroughly studied from an ecological perspective. For this tick-borne disease, a complex set of mechanisms involving the affect of fragmented forest—including that of forest edge habitat on the abundance the pathogen's most competent vector (white-footed deer mice)—on host and non-host mammal community abundance and composition explains disease emergence (LoGiudice et al., 2003). A number of other rodent-borne EIDs potentially can be explained by this or similar ecological mechanisms, although limited or no ecological research has been conducted. Rodents are well-known to frequently undergo what ecologists call "ecological release" when forest edge and clearings, as well as cropland, is created. In addition, human settlements provide abundant nesting, feeding, and breed sites for rodents and arthropods. Both groups have numerous species apparently pre-adapted to peri-domestic or domestic circumstances, often involving amplification of transmission via domestic animals. Thus arboviral EIDs such as Rift Valley fever, and Venezuelan Equine Encephalitis probably are ecotone associated. Nipah virus is a clearly documented example of amplification via agricultural encroachment into tropical forest; in this case for the dual purpose of establishing mango plantations and the raising of pigs (Olival and Daszak, 2005).

Sin Nombre virus is a rodent-borne (deer mouse) disease that was first recognized in the four corners region of the United States in the early 1990s. It caused a particularly virulent condition, hantavirus cardiopulmonary syndrome (HCPS) or hantavirus pulmonary syndrome (HPS), resulting in an initial case fatality rate of 80% (CDC, 1993). It is hypothesized that the initial emergence event followed El Nino-Southern oscillation (ENSO), causing increased precipitation that resulted in increased rodent

population densities and human interactions (Engelthaler et al., 1999). The majority of exposure is believed to occur indoors from contact with rodent excreta, at the interface of human settlement and natural ecosystem ecotone. A number of ecologically similar diseases, sharing rodent reservoirs and exposure, have also been recognized with increasing frequency worldwide and include Andes, Junin, Lanugan Negra, Lassa, Machupo, Puumala, Sabia, Seoul, Guanarito, Hantaan.

In areas in which livestock rearing is juxtaposed next to areas such as forests in which enzootic EIDs exist, the diseases may first spillover to domestic livestock and be amplified prior to additional spillover and emergence into human populations, such as in the case of human rabies outbreaks in northern Brazil (Gupta, 2005). Pigs may be involved as an intermediary step in a number of these instances including Nipah, Influenza, and Japanese Encephalitis. Spillover may also occur in both directions as illustrated by infectious agents as diverse as *Echinococcus granulosus*, *Bacillus anthracis*, *Mycobacterium bovis*, Rift Valley fever, Crimean-Congo hemorrhagic fever, and Kyasunur Forest virus. Animals congregate in ecotones, facilitating spillover. Possibly the best example is avian influenza, involving the mixing of three different communities: wild migratory waterfowl, wild local birds (and possible other vertebrates), and domestic fowl (chickens, geese, and ducks). In this case, all three of the above factors may combine to facilitate emergence. These affects may be further multiplied by the geographic convergence of aquatic-terrestrial ecotone with multiple types of human-created ecotones, further intensifying ecotonal processes. Nutrient pollution, degradation of riparian habitat with associated loss of ecological functions that normally assimilate nutrients and pathogens, combined with concentration of domestic fowl and their waste are commonly associated with human settlement/aquatic-terrestrial ecotones. Added to this is the tendency for human land use, particular that associated with urbanization, to increase the variety and amount of runoff along with more erratic hydrology (Allen, 2004). Virtually all of the waterborne EIDs arguably are at least in part associated with aquatic-terrestrial ecotonal processes, of which cholera and leptospirosis are well documented. Cholera (*V. cholerae*, *V. vulnificus*, and *V. parahaemolyticus*) is associated both with the land-ocean ecotone (estuary), since marine crustaceans are their natural reservoirs, and terrestrial-freshwater ecotones where human settlement and rivers or wetlands converge as describe above.

CONCLUSION

Ecotones have long been recognized as an important ecological setting favoring the transmission of a few well-described zoonotic infections, such as Yellow fever, where host or vector species congregate or otherwise occur in higher abundance than in either of the adjacent habitats. The ecotone concept as recently elaborated by landscape ecologists and the apparent correspondence with the biophysical and ecological evolutionary processes believed to generally underlie zoonotic EIDs may prove useful in better understanding the causes of the recently increasing global EID trend.

However, even for the 10 diseases identified here with a strong ecotone-association, ecotones may represent only one of several other important factors contributing to emergence. For example, human density, sanitation and hygiene, environmental quality, and pathogen dispersal, quite independent of ecotones and ecotonal processes, clearly contribute to the epidemiology of influenza, cholera, and malaria.

Also, the inclusiveness of landscape ecologists' expanded definition of ecotones and ecotonal processes, and the generality in terms of the association with disease emergence, arguably diminishes its predictive value. Specific ecotones will need to be more precisely defined, perhaps categorized or classified using landscape ecological methods, and such factors as intensity and concentration of the corresponding biophysical processes measured to develop predictive models of any practical utility based on the ecotone concept.

Yet ecotones apparently play a role in the some of the world's most important zoonotic EIDs. Current EID "hotspots" such as equatorial Africa and Southeast Asia seem to coincide with the pace of regional environmental change and its concomitants—urbanization, agricultural intensification, and habitat alteration (Wilcox and Colwell, 2005). That ecotones expand and ecotonal processes intensify with the creation and spread of multiple types of overlapping ecotones, as a result of and detectable by monitoring human activities such as land use, suggests the concept may be helpful in infectious disease research, surveillance, and prevention. The similar case made for their potential as sentinels for global climate change (Gosz, 1991, 1993; Allen and Breshears, 1998; Peteet, 2000) strengthens this argument.

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